(1) Modified lactic acid milk to which was added 2% casein hydrolysate.
(2) Dried half-cream lactified milk.

A gradual change from the first to the second stage was made at about the end of the second week. Analyses of these feeds gave almost identical figures, but 2% of the protein was hydrolysed in the first stage. There was no evidence of intolerance to the hydrolysed casein supplement.

Some cases had mild infections, but their progress was not thereby impeded. There was a steady gain in weight starting immediately after operation, averaging 6 oz. per week. This compares favourably with similar cases on lower protein feeds.

Blood was collected not less than 2 hr. after feeds at weekly intervals, starting 24–28 hr. after operation, when the patient was fully hydrated. The initial total serum proteins were within the normal range. There was a gradual fall in the total protein, albumin, globulin and haemoglobin followed by a rise. Albumin reached its lowest level approximately at the end of 2 weeks, globulin in 3 weeks and haemoglobin in 5 weeks.

Vitamin A in Rheumatic Fever. By Z. A. LEITNER, T. MOORE and I. M. SHARMAN, *Dunn Nutritional Laboratory, University of Cambridge and Medical Research Council*

Serial estimations of vitamin A and carotene were made on the blood plasma of 102 patients, mostly children, who suffered from rheumatic fever, in various stages of activity and convalescence. The mean values for all subjects for both vitamin A and carotene varied in an inverse direction to the body temperature and the erythrocyte sedimentation rate. The movements of these four variables, however, were not always co-ordinated in individual subjects. Similar decreases in vitamin A and carotene were found in fevers which were not of rheumatic origin. The possibility that repeated attacks of rheumatic fever may seriously impair vitamin A status was supported by a re-examination of previous data on the vitamin A reserves of children who had died from heart disease. Extremely low reserves were found in five cases of chronic or sub-acute rheumatic endocarditis.

The Second Ordinary Scientific Meeting of The Nutrition Society was held in the Barnes Hall of the Royal Society of Medicine, 1 Wimpole Street, London, W. 1, on Saturday, 31 May 1947, at 10.30 a.m., when the following papers were read:

The Intestine as a Possible Seat of Conversion of Carotene to Vitamin A in the Rat and the Pig. By S. Y. THOMPSON, J. GANGULY and S. K. KON, *National Institute for Research in Dairying, University of Reading*

Glover, Goodwin & Morton (1947) have just reported their findings on the conversion of carotene to vitamin A in the intestine of the rat. Independent work at Shinfield fully confirms these observations.

The accompanying table giving some of the results shows that in the fasted vitamin A deficient rat administration of carotene is followed within 30 min. by the appearance of vitamin A in the intestinal wall (and also in the contents) in quantities larger than in the liver or in the circulating blood. At 2 hr. relatively large quantities of vitamin A ester are present in the blood. Vitamin A was measured as described by Ganguly, Kon & Thompson (1947).

Vitamin A in the blood and organs of vitamin A deficient rats, after the administration of 2·6 mg. carotene in 10 drops of arachis oil

Rats fasted 16 hr. before dosing.

	Vitamin A i.u./rat				
	Blood plasma		Small intestine		
	Alcohol	Ester	Wall	Contents	Liver
Untreated rats	1·1	0·2	0·4	0·7	1·1
	0·4	0·2	0·4	0·4	0·5
Dosed ½ hr. before killing	0·7	0·3	3·6	4·5	1·3
	0·4	0·4	6·4	12·8	1·5
Dosed 1 hr. before killing	0·6	0·8	8·1	13·2	2·9
	0·6	0·6	5·1	7·7	4·4
Dosed 2 hr. before killing	2·7	0·7	4·7	18·0	14·3
	2·2	2·3	8·4	14·4	18·4

It appeared from other tests that more carotene was converted to vitamin A in the presence of food.

In bacon pigs dosed with 600 mg. β-carotene in arachis oil 3–7 hr. before slaughter, the vitamin A ester in blood plasma was increased and vitamin A was found in the mesenteric lymphatics and in the intestinal wall and contents in quantities much larger than in the control pigs.

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The Effect of Yeast on Liver Size and Vitamin A Storage in the Pig. By R. BRAUDE, K. M. HENRY, S. K. KON and S. Y. THOMPSON, *National Institute for Research in Dairying, University of Reading*

In the course of some experiments on yeast as a source of protein for pigs (Braude, Kon & White, 1943, 1944) the vitamin A content of the livers of the yeast-fed pigs proved lower than that of the control animals. A later examination of data of these and other experiments in which yeast was fed showed that the livers of yeast-fed pigs tended to be heavier than those of control animals.

In two specially planned experiments each with six pairs of Large White pigs, one pig of each pair received a normal fattening ration and the other the same ration with

14% dried brewer's yeast and 2% minerals replacing 10% fish meal and 6% of the barley meal, the vitamin A intake from cod-liver oil being the same in both groups. In one experiment the diets were given for 6 weeks and in the other for 22 weeks

Effect of yeast feeding on liver size and vitamin A storage in pigs

Diet	Dead wt. of pig (kg.)	Wt. of liver (kg.)	Liver wt. as % of dead wt.	Moisture in liver %	Nitrogen in liver* %	Fat in liver† %	Vitamin A in liver† i.u./g.	Vitamin A in liver† i.u./whole liver
Control (6 weeks)	71·82	1·41	1·97	70·77	—	1·20	101·5	144,000
Yeast (6 weeks)	71·37	1·53	2·14 $P=1:19$‡	70·98 N.S.	—	1·22 N.S.	63·3	96,000 $P=1:44$‡
Control (22 weeks)	80·74	1·45	1·80	70·97	3·41	0·83	118·0	171,000
Yeast (22 weeks)	78·24	1·62	2·08 $P=1:23$‡	70·74 N.S.	3·34 N.S.	0·90 N.S.	70·0	114,000 $P=1:30$‡

* By the macro-Kjeldahl method.
† By the extraction method of Davies (1933).
‡ The paired *t*-test of 'Student' (1908, 1925) was used. N.S. not significant.

before slaughtering at bacon weight. The results in the table show that the feeding of yeast caused an increase in liver weight not accounted for by differences in water or fat content, but that the storage of vitamin A in the liver was adversely affected.

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A Survey of Nutritional Deficiencies of Farm Crops in Somerset Using 'Visual' and 'Tissue Tests' Methods.

By W. PLANT, *Long Ashton Research Station*

During the last few years a field technique has been developed at Long Ashton for determining the nutrient condition of growing crops. A survey of the arable land of Somerset (167,000 acres) was carried out during the summer of 1946 in order to assess the incidence of deficiencies of the major elements, nitrogen, phosphorus, potassium and calcium, and of the trace elements, boron and manganese. A random farm sample representing 2% of all holdings was selected from farms of 10 or more acres. The following crops were inspected: cereals, beans, potatoes, sugar beet, roots and kale. The evaluation of crop vigour, which is based on empirical standards for each crop, was done visually. The nutrient condition of a crop was assessed by the use of two complementary methods, (*a*) 'visual' and (*b*) 'tissue-tests'. Technique (*a*) represents symptomatology and (*b*) colorimetric and turbidity tests used *in situ* on the crop. The season of 1946 was excessively wet and no periods of drought occurred.

The soils of Somerset are mostly medium to heavy loams. Only N, P, K and Ca deficiencies were recorded. N and P deficiencies were widespread; K deficiency was confined to potatoes and beans, and Ca deficiency to roots. The incidence of N deficiency in cereals was 15%; in potatoes it was also 15%; and in kale and roots 25%. The incidence of P deficiency in cereals was 21%; in potatoes it was 8% and in roots 2%. The incidence of K deficiency in potatoes was estimated at 15% and in beans at 20%; Ca deficiency was less than 1% in roots. Overall deficiencies for the county showed that 18% (30,000 acres) of the tillage land was deficient in N; 19% (31,000 acres) in P and 1% (2000 acres) in K. It is estimated that 16,000 acres of arable land would benefit from applications of nitrogen; 30,000 acres would benefit from dressings of phosphatic manures especially for cereal growing, and 2000 acres of 10,000 devoted to potatoes and beans require further dressings of potash fertilizers.

The Synthesis of Ascorbic Acid in the Vitamin A Deficient Rat. By L. W. MAPSON and SONIA E. WALKER, *Dunn Nutritional Laboratory, University of Cambridge and Medical Research Council*

Several workers (Sure, Theis & Harrelson, 1939; Boyer, Phillips, Pounden, Jensen, Rupel & Nesbit, 1942; Jonsson, Obel & Sjöberg, 1942, 1945) have shown that a deficiency of vitamin A causes a diminution in the ascorbic acid content of the tissues of rats and bovines. Some have claimed that the absence of vitamin A *per se* is responsible for an impairment in the synthesis of ascorbic acid. We have confirmed that the concentration of ascorbic acid in blood and liver but not in the suprarenals is lower in the vitamin A deficient than in the normal rat. This diminution in the ascorbic acid content of the body tissues is not due to increased urinary excretion, but can be accounted for by the lowered food intakes of the deficient animals.

Chloretone increases the synthesis of ascorbic acid in normal rats (Longenecker, Fricke & King, 1940; Smythe & King, 1942), and we have found that the urinary excretion of ascorbic acid and its concentration in the tissues of the vitamin A deficient animal is increased after ingestion of the drug. The rise in the urinary excretion of ascorbic acid in response to the drug is less marked in vitamin A deficient than in normal animals, but this difference is eliminated if the food intake of the controls is restricted to that of the deficient animals.

We have thus no evidence that vitamin A plays any specific role in the synthesis of vitamin C.

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